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Young Investigator Awards Competition

VAGUS NERVE STIMULATION INITIATING DURING ISCHEMIA, BUT NOT REPERFUSION, EXERTS CARDIOPROTECTION AND IS ASSOCIATED WITH AMELIORATION OF CARDIAC MITOCHONDRIAL DYSFUNCTION

Oral Contributions
Room 147 B
Sunday, March 30, 2014, 11:30 a.m.-11:45 a.m.

Session Title: Young Investigator Awards Competition: Physiology, Pharmacology, and Pathology
Abstract Category: Physiology, Pharmacology, Pathology
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Background: Initiation of left vagus nerve stimulation (VNS) prior to the onset of the ischemia provides cardioprotection against ischemia-reperfusion (I/R) injury. We sought to determine whether VNS initially applied during ischemia or at the onset of reperfusion exerts differential cardioprotection against I/R injury.

Methods: Pigs (25-30 kg, n=28) were randomized into 4 groups (Panel A). Ischemia was induced by LAD occlusion for 60 min followed by 120 min of reperfusion. VNS (3.5 mA, 20 Hz, continuously recurring cycles of 21-s ON, 30-s OFF) was initiated either 30 min after ischemia onset, or at the reperfusion onset, and continued until the end of reperfusion. The ischemic and non-ischemic myocardium was harvested for cardiac mitochondrial function assessment.

Results: VNS initiated 30 min after LAD occlusion, but not at reperfusion, markedly reduced infarct size (~59%) and VF episodes, improved LV function, and attenuated cardiac mitochondrial ROS production, depolarization, swelling and cytochrome c release, compared to Control (Panel B-E). These beneficial effects of VNS were completely abolished by Atropine (1mg/kg), suggesting a strong influence of a cholinergic-dependent pathway.

Conclusion: VNS provides significant cardioprotective effects when initiated during ischemia, but not during reperfusion period. These findings indicate the importance of timing for VNS initiation, and warrant the potential clinical application of VNS in protecting myocardium at risk of I/R injury.